

<b>INFORMATION DISCLOSURE CITIGATION IN AN APPLICATION</b>  (Use several sheets if necessary)			Attorney Docket No.:	Application No.:		
			<b>1510-1121</b>	<b>10/593,639</b>		
			Applicant: <b>Lars NILSSON et al.</b>			
			Filing Date:	Group Art Unit:		
			<b>September 21, 2006</b>	<b>1632</b>		
<b>U.S. PATENT DOCUMENTS</b>						
Examiner Initial	Document Number	Date	Name	Class	Subclass	Filing date (if appropriate)
<b>FOREIGN PATENT DOCUMENTS</b>						
Examiner Initial	Document Number	Date	Country	Class	Subclass	Translation Yes No
/D.C./	WO 02/003911	01-17-2002	WIPO			
/D.C./	WO 02/102412	12-27-2002	WIPO			
/D.C./	WO 04/041213	05-21-2004	WIPO			
<b>OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)</b>						
/D.C./	CAI et al., "Release of excess amyloid b protein from a mutant amyloid b protein precursor," <i>Science</i> , Vol. 259, No. 5094, January 22, 1993, pp. 514-516.					
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EXAMINER:			DATE CONSIDERED		09/30/2008	
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<i>/D.C./</i>	<p>KANG et al., "Letters to <i>Nature</i>: The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor," <i>Nature</i>, Vol. 325, February 19, 1987, pp. 733-736.</p> <p>KLYUBIN et al., "Soluble arctic amyloid B protein inhibits hippocampal long-term potentiation in vitro," <i>European Journal of Neuroscience</i>, Vol. 19, 2004, pp. 2839-2846.</p> <p>LANTOS et al., "Familial Alzheimer's disease with the amyloid precursor protein position 717 mutation and sporadic Alzheimer's disease have the same cytoskeletal pathology," <i>Neuroscience Letters</i>, Vol. 137, 1992, pp. 221-224.</p> <p>LASHUEL et al., "Mixtures of wild-type and a pathogenic (E22G) form of A B40 <i>in vitro</i> accumulate protofibrils, including amyloid pores," <i>Journal of Molecular Biology</i>, 2003, Vol. 332, pp. 795-808.</p> <p>LI et al., "Intracellular accumulation of detergent-soluble amyloidogenic A B fragment of Alzheimer's disease precursor protein in the hippocampus of aged transgenic mice," <i>Journal of Neurobiology</i>, 1999, pp. 2479-2487.</p> <p>LORENZO et al., "B-amyloid neurotoxicity requires fibril formation and is inhibited by Congo red," <i>Proceedings of the National Academy of Science USA</i>, Vol. 91, December 1994, pp. 12243-12247.</p> <p>MASTERS et al., "Amyloid plaque core protein in Alzheimer disease and Down Syndrome," <i>Proceedings of the National Academy of Sciences USA</i>, Vol. 82, June 1985, pp. 4245-4249.</p> <p>MULLAN et al., "A pathogenic mutation for probable Alzheimer's disease in the APP gene at the N-terminus of <math>\beta</math>-amyloid," <i>Nature Genetics</i>, Vol. 1, August 1992, pp. 345-347.</p> <p>MURRELL et al., "A mutation in the amyloid precursor protein associated with hereditary Alzheimer's disease," <i>Science</i>, Vol. 254, No. 5028, October 4, 1991, pp. 97-99.</p> <p>NASLUND et al., "Correlation between elevated levels of amyloid <math>\beta</math>-peptide in the brain and cognitive decline," <i>Journal of the American Medical Association</i>, Vol. 283, No. 12, March 22/29, 2000, pp. 1571-1577.</p> <p>NILSBERTH et al., "The 'Arctic' APP mutation (E693G) causes Alzheimer's disease by enhanced <math>\beta</math>B protofibril formation," <i>Neuroscience</i>, Vol. 4, No. 9, September 2001, pp. 887-893.</p> <p>NILSSON et al., "<math>\alpha</math>-1-Antichymotrypsin promotes <math>\beta</math>-sheet amyloid plaque deposition in a transgenic mouse model of Alzheimer's disease," <i>The Journal of Neuroscience</i>, Vol. 21, No. 5, March 1, 2001, pp. 1444-1451.</p> <p>PIKE et al., "In vitro aging of <math>\beta</math>-amyloid protein causes peptide aggregation and neurotoxicity," <i>Brain Research</i>, Vol. 563, 1991, pp. 311-314.</p> <p>ROHER et al., "The human amyloid-<math>\beta</math> precursor protein<sup>770</sup> mutation V717F generates peptides longer than amyloid-<math>\beta</math>-(40-42) and flocculent amyloid aggregates," <i>The Journal of Biological Chemistry</i>, Vol. 279, No. 7, February 13, 2004, pp. 5829-5836.</p> <p>SCHEUNER et al., "Secreted amyloid <math>\beta</math>-protein similar to that in the senile plaques of Alzheimer's disease is increased <i>in vivo</i> by the presenilin 1 and 2 and APP mutations linked to familial Alzheimer's disease," <i>Nature Medicine</i>, Vol. 2, No. 8, August 1996, pp. 864-870.</p> <p>SELKOE, "Cell biology of the <math>\beta</math>-amyloid protein precursor and the mechanism of Alzheimer's disease," <i>Annual Review of Cell Biology</i>, Vol. 10, 1994, pp. 373-403.</p> <p>SELKOE, "Normal and abnormal biology of the <math>\beta</math>-amyloid precursor protein," <i>Annual Review of Neuroscience</i>, Vol. 17, 1994, pp. 489-517.</p> <p>STENH et al., "The Arctic mutation interferes with processing of the amyloid protein precursor," <i>NeuroReport</i>, Vol. 13, No. 15, October 28, 2002, pp. 1857-1860.</p> <p>STRITTMATTER et al., "Apolipoprotein E: High-avidity binding to <math>\beta</math>-amyloid and increased frequency of Type 4 allele in late-onset familial Alzheimer disease," <i>Proceedings of the National Academy of Science USA</i>, Vol. 90, March 1993, pp. 1977-1981.</p> <p>STURCHLER-PIERRAT et al., "Two amyloid precursor protein transgenic mouse models with Alzheimer disease-like pathology," <i>Proceedings of the National Academy of Science USA</i>, <i>Neurobiology</i>, Vol. 94, November 1997, pp. 13287-13292.</p> <p>SUZUKI et al., "An increased percentage of long amyloid <math>\beta</math> protein secreted by familial amyloid <math>\beta</math> protein precursor (baPP<math>\beta</math> (717)<math>\delta</math>) Mutants," <i>Science</i>, Vol. 264, No. 5163, May 27, 1994, pp. 1336-1340.</p> <p>WALSH et al., "Letters to <i>Nature</i>: Naturally secreted oligomers of amyloid <math>\beta</math> protein potently inhibit hippocampal long-term potentiation <i>in vivo</i>," <i>Nature</i>, Vol. 416, April 4, 2002, pp. 535-539.</p>		
<i>/D.C./</i>	EXAMINER: <i>Deborah Crouch</i>	DATE CONSIDERED <i>09/30/2008</i>	
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